Skeletal Muscle Force

Introduction

Skeletal muscle shortens to bring two bones closer together. Some muscles are big, some are small. But all muscles obey the same general concepts when it comes to the generation of force. "How hard you try to lift something" is translated into recruitment of fibers, summation of current fibers, and also a little stretch of elastic connective tissue. In this lesson we look at the stretch-force relationship of sarcomeres isolated from a living organism, then apply what we've learned about excitable cells to talk about what actually happens when someone pumps iron in the gym. We start with actin/myosin overlap (sarcomeres), move to neurons (summation and recruitment), then talk about what happens in actual muscles that are inside a human (active and passive tension). We finish with the twitch types and observations on both.

Active Tension from a Sarcomere = Actin/Myosin Overlap

The force an individual sarcomere can generate is dependent on the overlap of actin and myosin. It's the sliding of actin past myosin that causes a sarcomere (and therefore the muscle) to shorten. The more overlap, the more heads are available to participate in the powerstroke. Follow along with Figure 14.1.

The first thought is to maximize overlap of myosin and actin. But remember, the sarcomere has to shorten. If there were maximal actin and myosin overlap, then actin would already be near the M line and have nowhere to move. Consider how ridiculous it would be to attempt a curl when already in the finished, contracted position. The arm would have nowhere to move. The same is true for the sarcomere. **Too much overlap** cannot generate any shortening (position A).

Going to the opposite extreme, if there's **no overlap at all** there could be no initiation of the powerstroke (point C). This stretch represented by point C is beyond physiologically possible, but it gets the point across: no overlap, no powerstrokes, no contractile force.

And as we move in from the extremes, the active tension that can be developed from a sarcomere increases. There's a **sweet spot** of **maximal actin and myosin overlap** but still **sufficient room to shorten**. This occurs when actin covers all of the myosin heads, but does not cover the bare zone (point B).

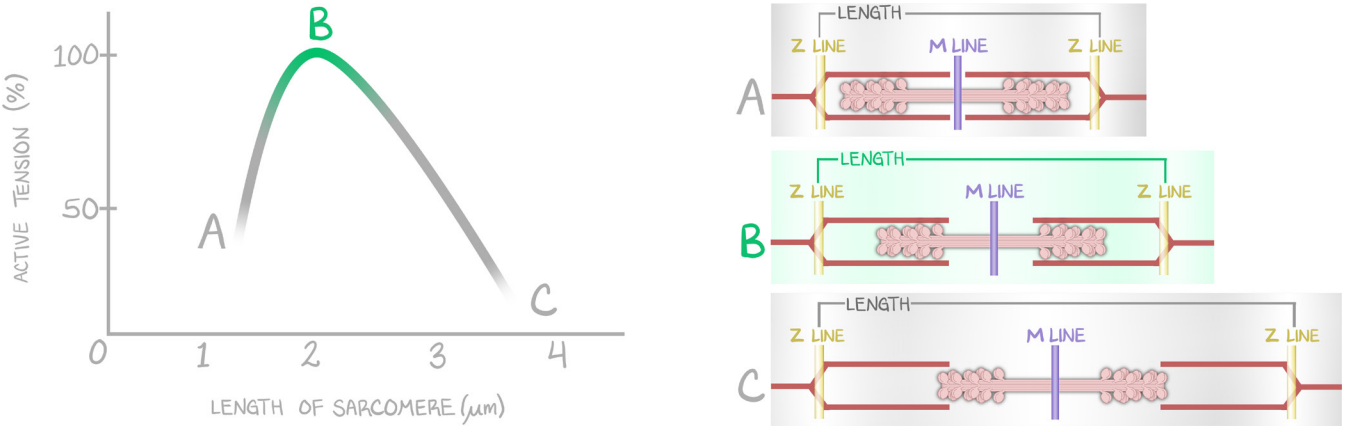

Figure 14.1: Length Tension of a Sarcomere
The force generated by any individual sarcomere is dependent on the overlap of actin and myosin. Active tension is optimized by the starting length, B. Too little overlap and there is nothing to generate the force, A. Too much overlap and there is nowhere for the actin filaments to go, C.

Active Tension from Whole Muscle: Summation and Recruitment

Students have a lot of problems with these concepts because this isn't what we do when we want to lift something heavy. We simply "try harder" or "give more effort." Our conscious will, our mental preparation, knows we have to lift harder against heavy weight. But what your neurons do, what actually happens in your spinal cord, end plate, and skeletal muscles, you have no knowledge of. Summation and recruitment are happening despite your inability to perceive them.

Summation occurs within a motor unit—repeated depolarization adds together to result in a stronger contraction of that motor unit. **Recruitment** happens sequentially—first the smaller motor units acting, then subsequently larger and larger motor units.

A **motor unit** is a collection of **muscle fibers** that are **innervated by the same neuron**. One neuron, many fibers. A motor unit can be **large** (many fibers for one neuron) in **bigger muscles** that do not require fine motor control. A motor unit can be **small** (few fibers for one neuron) in small agile muscles like the fingers, eye, or larynx. Regardless, there are multiple motor units in each gross muscle. Your biceps is not all one motor unit; it's made of multiple. And motor units **are not consecutive fibers**. Motor units overlap physically to permit summation and recruitment and to control the contraction of the muscle trajectory.

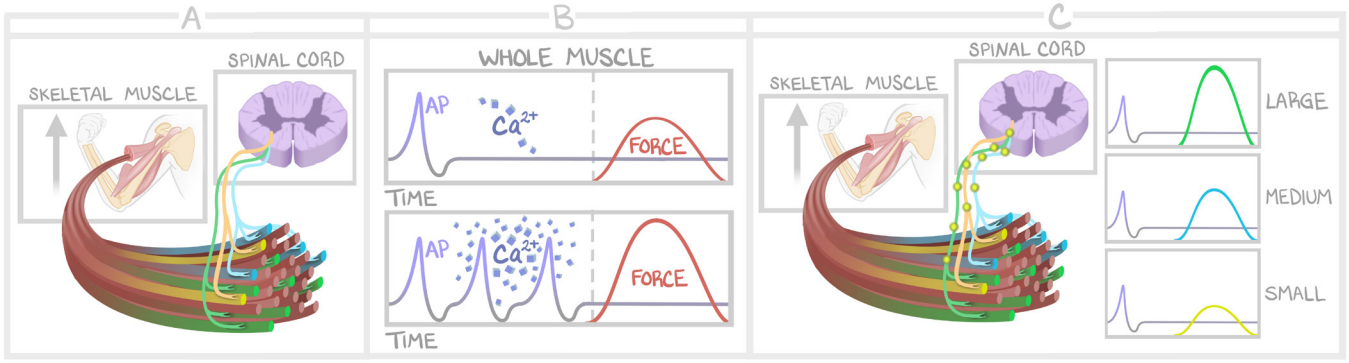

Figure 14.2: Motor Units, Summation, and Recruitment
(a) A motor unit is a single motor neuron and all of the noncontiguous muscle fibers it innervates. (b) Within a single motor unit, repeated action potentials of the motor neuron may occur before contraction begins, each depolarization injecting more calcium into the cytoplasm of the skeletal muscle, summating the contractile force of multiple depolarizations in one contraction. (c) Subsequent motor fibers, each progressively larger than the last, are added to the contraction with recruitment.

When you don't try very hard to lift something, your brain sends a weak stimulus to move. A weak stimulus can activate only the most excitable neurons. The most excitable neurons are the **smallest** and **innervate the smallest motor unit**. When you try really hard to lift something, especially if you cannot actually move it but you keep trying, your brain sends many signals, strong ones, over and over again. This causes the **activation of multiple motor units** (recruitment) and **summation within motor units.** Since the smallest, weakest motor units are added first, as more motor units are added, there's a stepwise and exponential increase in force.

The **action potential** that initiates a contraction is of immensely short duration relative to the duration of contraction. The action potential is started and ended before calcium levels rise. The calcium levels are already being sequestered when the force of the contraction is felt. Therefore, if **additional action potentials are added** even before the force of contraction begins, the additional calcium influx would increase the force of contraction. This is **temporal summation**. Continuing to stimulate the endplate gets a stepwise increase in the force of contraction until **tetany** is reached, where all the motor units are fully contracted. More stimuli simply cannot add any more calcium to the sarcomere.

Finally, the actual work that a whole muscle can do is limited below the sum of all of its sarcomeres. In order to lift a weight AND hold it up, there must be **asynchronous** activation of muscle fibers. If all the myosin contracted at once (synchronous), you would get a more powerful contraction, but then the moment they synchronously detached, there would be nothing to sustain the contraction.

Real Muscles: Active and Passive Tension

Active tension at the level of the sarcomere has to do with actin and myosin overlap, which translates to whole muscle. Whole muscle could use temporal summation and motor unit recruitment to generate more force, and theoretically activate all the fibers at once, matching the actin-myosin curve of the sarcomere. However, asynchronous firing of the motor units is required to sustain the contraction, so the whole muscle could never reach the ideal sum of all sarcomeres together.

But real muscle isn't just a combination of many fibers together. There's also **connective tissue** that surrounds it. Connective tissue is **elastic**—it snaps back and doesn't like being stretched. It's like a rubber band—at rest, it's happy the way it is. Stretch it, and it wants to snap back, and that "desire to snap back" to where it started is **passive tension**. To prove it to yourself, stand up and touch your toes without bending your knees. Feel that "stretch" in your hamstrings? Yep. That's the initial force of that elasticity, just a touch of passive tension of your hammies telling you they want to go back to being not stretched.

There should be **almost no passive tension** on a human's skeletal muscle. Generating sufficient stretch of a muscle to have the elasticity generate a noticeable force of passive tension would stretch the muscle beyond physiology's reach. That tightness in your hamstrings doesn't snap you back upright. When you do biceps curls, and you lower your arm to the most extended position, your range of motion is limited by the elbow joint (the elbow locks at 180 degrees). That degree of biceps stretch is insignificant in the generation of a force of contraction because the elbow locks before the biceps is stretched long enough for the connective tissue to add elastic recoil. If a biceps muscle is removed from a human and stretched without that limitation, very quickly after physiologic lengths, the elastic tension grows rapidly.

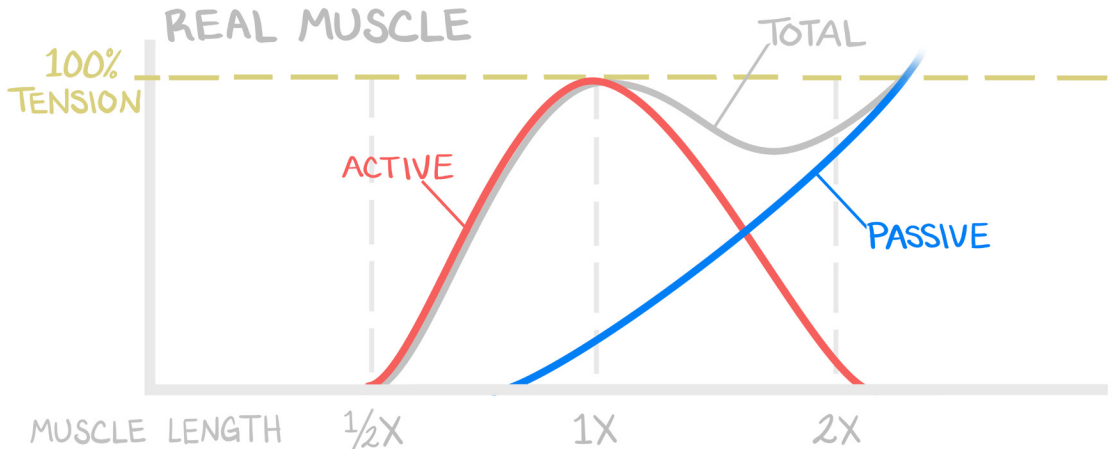

Figure 14.3: Active, Passive, and Total Tension
Active tension is generated by sarcomeres, passive tension by the stretch of connective tissue. At shortened muscle lengths there is no stretch on the connective tissue, and while there is good overlap of actin and myosin, there is nowhere for the actin to move (1/2x). No passive tension, no active tension, no total tension. As skeletal muscle is stretched to the physiologic length (1x), the sarcomere alignment improves, yet there is still no stretch on connective tissue. Therefore, the active tension mirrors total tension. As the muscle gets really stretched (2x), the connective tissue resists that stretch, generating passive tension. The sarcomeres get overstretched, so diminish active tension, yet total tension increases. To the right of 2x there is so much passive tension that the tendon snaps.

In real muscles we can assess the total force of contraction relative to the starting length of the muscle. At **physiological levels, there's** nearly **no passive tension**, and the muscle can achieve **maximal effort**. If the muscle is stretched to pathological levels, the **total force** of contraction initially doesn't change—the **lost actin and myosin overlap** is replaced by the **passive tension** from the elasticity of the tissue. Once lengths extend past impossible-to-achieve-in-humans (like twice that of a normal skeletal muscle), it becomes apparent that the loss of actin-myosin overlap means the total force that can be generated fails. If a human muscle is stretched to twice its length, it's likely simply to tear.

Isotonic and Isometric Contractions

Isotonic means the weight stays the same, and the muscle shortens. **Isometric** means the muscle doesn't shorten, though the load increases. Isometric contractions occur when there's insufficient force generated to move the load. The distinction between isotonic and isometric formed the basis for studies on the relationship between action potential and contraction of muscles of various size and length. From this we learned that there are **fast-twitch muscles** and **slow-twitch muscles**.

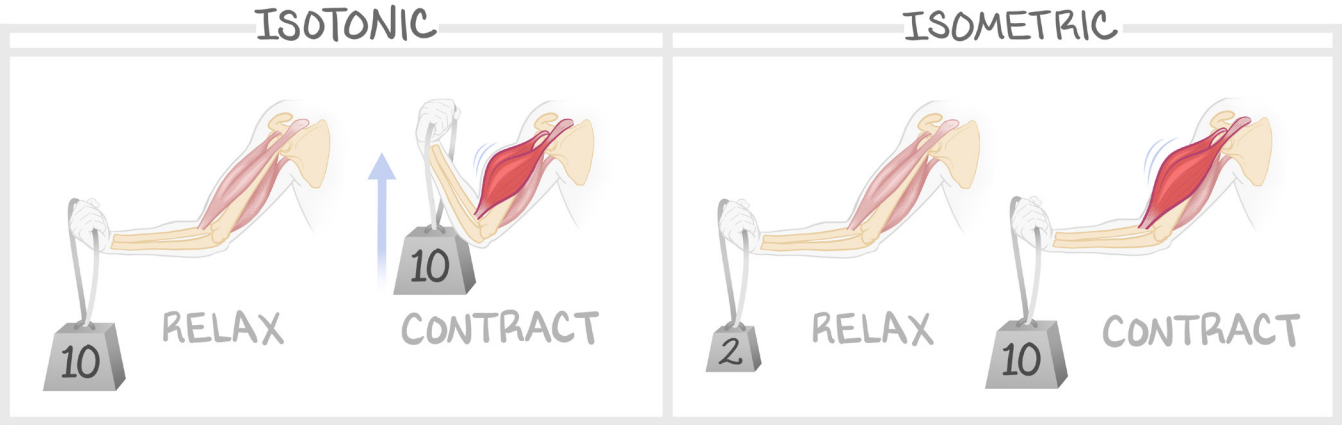

Figure 14.4: Isometric and Isotonic Contractions
Isometric contractions show no movement of muscle despite increased force. Isotonic contractions keep a constant load but the muscle shortens or lengthens.

Fast- and Slow-Twitch Muscles

Slow-twitch muscles are classified as **type 1** fibers. These are the fibers that can provide sustained contractions. These are postural muscles that enable us to remain upright while standing. They rely on **oxidative phosphorylation** for energy, do **not fatigue easily** (under aerobic conditions), and are rich with **myoglobin**, an oxygen-binding protein—myoglobin grabs onto oxygen and saves it for later. Myoglobin is to oxygen as glycogen is to glucose. Because they are so reliant on oxidative phosphorylation, they are **rich in mitochondria** and have **many capillaries** (oxygen for those mitochondria). The abundance of myoglobin and capillaries gives slow-twitch muscles a **red appearance**. Because contractions require ATP and because these muscles are almost always contracting, **their glycogen stores are often depleted**. Slow-twitch muscles have a **large motor unit** (one neuron, many muscle fibers).

Fast-twitch muscles are classified as **type 2b** fibers, fast-fatigable. Fast-twitch muscles are those that are used for rapid, coordinated movements, such as moving the eye. Type 2b appear **white** because they lack mitochondria and capillaries. Because coordination and agility are granted by more neurons and fewer muscles per neuron, fast-twitch muscles often require a **small motor unit** (one neuron, few muscle fibers). They sit around, not contracting, then when they need to contract, they do it in a **burst of activity**. This burst nature means they have downtime to **store glycogen**. When they contract, they

will use it all, but they are not invested in complex cellular respiration—they mostly gain their ATP from glycolysis rather than from TCA-ETC. Fast-twitch, because they use glycolysis only (effectively anaerobic metabolism), and **fatigue quickly** from the accumulation of lactate.

CHARACTERISTIC	SLOW-RESISTANT TYPE 1	FAST-FATIGABLE TYPE 2B
Primary source of ATP Mitochondria Capillaries	**Oxidative phosphorylation** Many Many	**Glycolysis** Few Few
Myoglobin content Glycolytic enzyme activity Glycogen content	**High** Low Low	**Low** High High
Rate of fatigue contract velocity	**Slow** **Slow**	**Fast** **Fast**
Fiber diameter Motor unit size Size of innervating neuron	Small Small Small	Large Large Large

Table 14.1: Slow-Twitch vs. Fast-Twitch Fibers
Comparison of slow-twitch red fibers and fast-twitch white fibers.